

# THE DEVELOPMENT OF ACUTE MYOCARDIAL INFARCTION DUE TO COMPRESSION OF THE LEFT CORONARY ARTERY TRUNK BY THE CARDIAC TUMOR

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**ABSTRACT** We report the clinical observation of acute myocardial infarction development associated with the compression of the left coronary artery trunk and discuss etiological role of primary and secondary cardiac tumors in the development of acute coronary syndrome.

**Keywords:** heart tumor, malignant fibrous histiocytoma, acute myocardial infarction, acute coronary syndrome

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ACS — acute coronary syndrome

AMI — acute myocardial infarction

CA — coronary artery

ECG — electrocardiogram

MFH — malignant fibrous histiocytoma

Atherosclerosis of coronary arteries (CA) obviously becomes the most common cause of severe coronary artery disorder and development of acute myocardial infarction (AMI), complicated by formation of an intravascular thrombus [1]. At the same time, external compression of CA is a rare cause in the development of acute coronary syndrome (ACS) and is observed in less than 0.1% of ACS cases. External compression of CA may be associated with various causes, including primary or metastatic tumors of myocardium or pericardium [2, 3], pulmonary artery dilatation in patients with significant pulmonary arterial hypertension [4-6].

Primary heart tumors are less common than secondary tumors. According to autopsy data, the incidence of primary heart tumors is 0.0001-0.0003% [7]. Secondary heart tumors in most cases are associated with lung cancer [8, 9]. Metastases in the heart are revealed in 25-30% of pathoanatomical studies of deceased patients who had primary lung cancer [9]. But during life such metastases are difficult to diagnose, because often they do not have clinical manifestations. Metastases in the heart are most often found in the pericardium and rarely in the myocardium or endocardium [10].

Among primary heart tumors, benign and malignant are 75% and 25%, respectively, with sarcomas in 75% of cases [11]. Malignant fibrous histiocytoma (MFH) is the second most frequent malignant tumor of the heart in adults. There is no primary development of MFH, depending on gender. The average age of patients with MFH is about 45 years. MFH is more often located in the left atrium in the area of its posterior wall and/or the interatrial septum [12-14]. According to the analysis of 47 cases of heart failure, in 81% of cases the tumor was located in the left atrium [15]. The other locations were pericardial cavity, right ventricle/pulmonary artery valve, right atrium and left ventricle were in 3, 3, 1 and 1 cases, respectively. The local growth of the tumor can lead to the development disorders of myocardial conduction or coronary circulation with the development of chest pain, AMI, heart block or sudden death.

Despite the fact that in secondary heart cancer (caused most often by lung cancer) with intramural tumor growth or metastasis to the myocardium, there may be changes in the ST segment on an electrocardiogram (ECG) resembling an STEMI picture that can be recorded even in the absence of CA occlusion [10]. However, there are only isolated descriptions of cases tumor compressed coronary arteries or invaded CA leading to its occlusion [2, 16]. In the literature, we did not find a description of AMI, associated with cardiac MFH. We report our own clinical observation of AMI development caused by external compression of the left coronary artery with MFH.

## Clinical observation

A 47-year-old male patient P. arrived via ambulance to the intensive care unit of the Cardiology Department No 3 of N.V. Sklifosovsky Research Institute for Emergency Medicine in agony 1 hour after deterioration of state of health with the diagnosis: acute myocardial infarction of the left ventricle anterior wall.

According to relatives, before the development of the clinical picture of the disease, the patient was tolerable to physical exertion, did not complain of pain in the chest and overall felt satisfactory.

Against the background of complete well-being, the patient suddenly complained of a feeling of lack of air and lost consciousness. There was a cold sweat. The arterial pressure recorded by the ambulance team was 60/30 mm Hg. The patient regained consciousness after dopamine infusion and complained of heaviness in his chest, asphyxiation.

On the ECG, registered by the ambulance team, there was an idioventricular rhythm, extended deformed QRS complexes, the frequency of ventricles contraction was 60-100 per min (Fig. 1). At the prehospital stage, the patient was given morphine 20 mg intravenously, simultaneous aspirin 300 mg and clopidogrel 300 mg. The tendency to arterial

hypotension persisted, despite the ongoing therapy. During transportation in the ambulance the patient repeatedly lost consciousness, the pulse on the main arteries was undetectable.

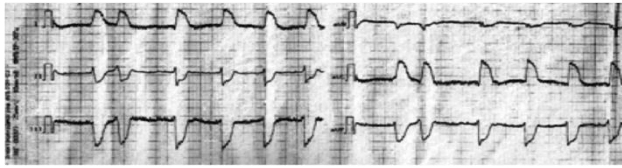


Fig. 1. The electrocardiogram registered by an ambulance team. Idioventricular rhythm

The patient arrived to the intensive care ward unconscious. Pupils OD = OS, expanded, no photoreaction. Diffuse muscular atony. Areflexia. Diffuse cyanosis with a gray tint. Agonal breaths with a frequency of 4-6 per minute. Heart sounds were not auscultated. Pulse on the main arteries was undetectable. The monitor of the defibrillator and ECG showed coarse ventricular fibrillation (Fig. 2).

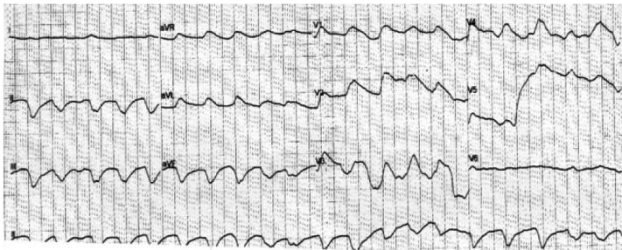


Fig. 2. The electrocardiogram registered in the intensive care ward. Coarse ventricular fibrillation

Resuscitation measures were immediately initiated. Artificial lung ventilation was started. Indirect cardiac massage, electroimpulse therapy were performed, a trunk vein was catheterized and a temporary endocardial pacemaker was installed. Resuscitation took 50 minutes had no effect. Biological death was pronounced.

Additional methods of examination, including coronarography, were impossible due to the terminal condition of the patient.

The pathoanatomical study revealed tumor in the left atrium compressing and externally occluding the lumen of the proximal part of the left coronary artery (Fig. 3), which caused the development of the left ventricular AMI. The lesion of the left ventricle of the heart was 30%.

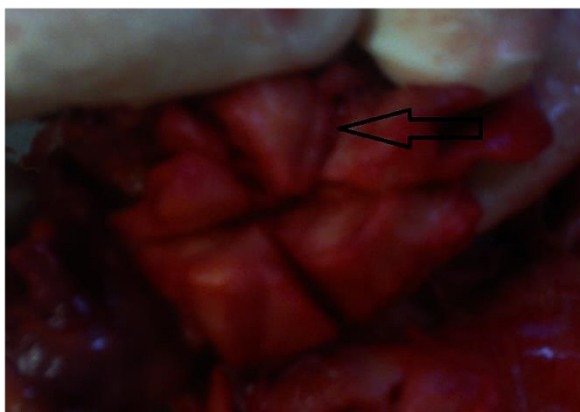


Fig. 3. Malignant fibrous histiocytoma of the left atrium with compression of the lumen of the proximal left coronary artery. Gross specimen

Pathologoanatomical diagnosis (after receiving the data of histological examination): malignant fibrous histiocytoma with infiltrative growth in the myocardium and pericardium, occlusion of the left coronary artery.

The death of a 47-year-old patient suffering from a malignant histiocytoma with occlusion of the left coronary artery was associated with acute myocardial infarction with events acute cardiac failure.

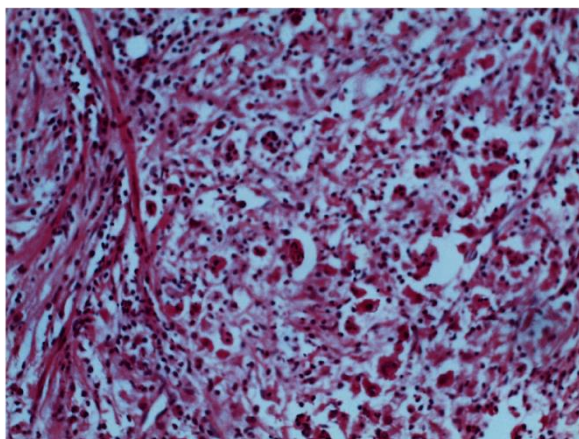


Fig. 4. Fibrous histiocytoma of the left atrium. Staining with hematoxylin and eosin, magnification 200x

## CONCLUSION

Thus, a rare case of AMI development due to occlusion of the left coronary artery of the MFH is presented. The absence of clinical manifestations of compression of the left main trunk before the development of AMI can be attributed to the peculiarities of the case. Despite the fact that heart tumors rarely become the direct cause of ACS, it should be remembered that coronary heart disease can not be considered the only cause of myocardial infarction.

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